THE SODIUM STATUS OF THE BODY IN RELATION TO SURGICAL STRESS AND INFUSION

THESIS

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C E R T I F I C A T E

This is to certify that the work entitled as " THE SODIUM STATUS OF BODY IN RELATION TO SURGICAL STRESS AND INFUSION " which is being submitted as Thesis for M.S.(General Surgery) examination, 1992 of Bundelkhand University, Jhansi has been carried out by Dr. SURENDRA DUTT SAKLANI Himself in this department.

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This is to certify that Dr.Surendra Dutt Saklani has worked on " The sodium status of body in relation to surgical stress & infusion" under my guidance and supervision.

His results and observation have been checked and verified by me from time to time.

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Dated: (Surendra Dutt Saklani)

CONTENTS

Page No.

INTRODUCTION

INTRODUCTION

Serum electrolytes mainly sodium and potassium are an intigral pact of the milieu interior and disturbance in their relative concentration occurs with stress, which may be as a result of either accidental or surgical trauma. Thus optimal management of surgical patient to-day demands a thorough knowledge of the changes in fluid and electrolyte balance associated with the surgical procedures. The reasons of these changes are however, far less completely understood. Changes in various serum electrolytes leads to various manifestations which are confused with the effect of anaesthesia or any other disease. But with newer diagnostic methods the alteration in serum electrolytes can be quantitated were accurately and treated accordingly.

Any surgical process or trauma leads to various body reactions. Acute changes in renal function in the operative & post operative period have been observed for many years and the decreased renal excretion of sodium in the post operative period, is well documented since a long time. Despite this fact

there may be a tendency towards hyponatremia in the early postoperative periods. It has been recently established that the intracellular fluid volume or more precisely the " functional extracellular fluid volume is a major determinants of the renal sodium excretion.

It was demonstrated that the metabolic responses of the body to surgical stress profoundly alters the need for salt and water in the post operative period. As a result of these studies it has been costomary for same years to limit the amounts of water and sodium administered in the postoperative period. Most clinicians believe that such limitation reducess the incidence of edema, dilutional hyponatremia and water intoxication, problems which have been noted to occur in the postoperative patient. A number of recent reports are indicative that there is an acute deficit in the volume of intracellular fluid space during surgical trauma and that a large amount of fluid is needed to correct this deficit. Also despite this fact that there is sodium retention after any kind of trauma, including surgery, in the early post operative period, there is still a tendency towards hyponatremia probably because of an associated fluid retention. The isotonicity of body fluids which is maintained between intracellular

and extracellular fluid, further increases hyponatremia, which occurs after surgical process. The implication of this hyponatremia, when seen in the light of the type of fluid administration per operatively and post operatively and effect of this fluid on the serum sodium level, is important for the clinician. In this context we needed to re-examine the usual concept of administration of sodium free fluid in the immediate post operative periods.

AIMS OF STUDY

1. To document the sodium and potassium status pre operatively and to see the changes in the serum sodium and potassium in early post operative periods.

2. To assess the effect of type of fluid given in the peroperative and early post operative period on serum osmolality.

3. To assess the effect of type of fluid given in the peroperative and early post operative period on urinary sodium and urinary volume.

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REVIEW OF LITERATURE

REVIEW OF LITERATURE

One of the most critical aspects of surgical patient's care relates to the management of fluid and electrolytes, especially because the operative trauma imposes a great impact on the physiology of body fluids and electrolytes.

Anatomy of body fluid :-

The body is composed of two major components

1. Non aqueous
2. Aqueous

Body fat and extracellular solids such as bone, tendon, fascia and collagen make up the non hydrous portion. The aqueous phase is in general the sum of three components extracellular water, blood volume and intracellular fluid.

Total body water

Water constitutes about 50-70% of total body weight using deuterium oxide or triatiated water for measurement of total body water (Moore et al 1963) have shown that total body water as a percentage of total body weight, decreases steadily and significantly with age to a low of 52 and 47% in males and females respectively. Since fat contains little

water, the lean individual has a greater proportion of water to total body weight than the obese person. Since females have greater quantity of fat so the percentage of water is less in females than of males of same weight.

Conversely, the highest proportion of total body water is found in newborn, which after several months after birth decreases gradually is a physiological loss as the child adjusts to the environment.

The water of body is divided into three functional compartments. The fluid with in the body's diverse cell population represents between 30-40% of body weight. The extracellular water represents approximately 20% of body weight and is divided between intravascular fluid, or plasma (5%) and interstitial or extravascular, extracellular fluid (15%).

<u>Young adult</u>

Plasma Interstitial fluid	Total extracellular volume 20% (Plasma 5%, Interstitial 15%)
Intracellular volume	Total intracellular volume 40%

<u>Intracellular fluid</u>

The intracellular water is between 30-40% of the body weight, The chemical composition of

intracellular fluid is given in below.

The potassium and magnesium are the principal cations and phosphates & proteins the principal anion in intracellular compartment.

Extracellular fluid

The total extracellular fluid volume represents 20% of body weight. The extracellular fluid compartment has two major subdivisions.

The plasma volume is approximately 5% of the weight of normal adult. The interstitial or extravascular, fluid volume obtained by substracting the plasma volume from the measured total extracellular fluid volume constitutes approximately 15% of body weight.

The interstitial, fluid is further complicated by having, normally, a rapidly, equilibrating or functional component, as well as several slowly equilibrating, or relatively nonfunctioning components The non functioning components include connective tissue water, as well as water that has been termed transcellular which includes cerebrospinal and joint fluids. This non functional component normally represents only 10% of the interstitial fluid volume (1-2%) and is not to be confused with the relatively nonfunctional extracellular fluid often labelled as third space found in burn & soft tissues.

The normal constituents of the three compartments are given below.

Plasma: Cation	Plasma: Anions	Interstitial fluid: Cation	Interstitial fluid: Anion	Intracellular fluid: Cation	Intracellular fluid: Anion
		153	153	200 Meq/L	200Meq/L
Na^+ 142	Cl^- 103	Na^+ 144	Cl^- 144	K^+ 150	HPo_4^{-3}, So_4 150
K^+ 4	Hco_3^{-3} 27	K^+ 4	Hco_3^{-3} 30	Mg^{++} 40	Hco_3^{-3} 10
Ca^{++} 5	So_4^{-2}, Po_4^{-2} 3	Ca^{++} 3	So_4^{-2} 3	Na^+ 10	Proteins 40
Mg^{++} 3	Org. ac. 5	Na^{++} 2	Po_4		
	Protein 16		Org. ac- 3		
			Proteins 1		
154 Meq/L	154 Meq/L				

Plasma   Interstitial fluid   Intracellular fluid

The sodium is major cation of the extracellular fluid & contributes more than 90% to the osmolality of this compartment.

Osmotic pressure & osmolality

The osmolality of body fluids is defined as " the osmolar concentration expressed as osmols/litre of solution. Osmoles is the number of osmotically active particles or ions per units volume".

The difference in ionic composition of intracellular & extracellular fluid compartments are maintained by the cell wall, which functions as semipermeable membrane. The osmotic pressure of fluid is the sum of the partial pressure contibuted by each of the solutes in that fluid, the effective osmotic pressure is dependent on those substance that fail to pass through pores of semipermeable membrane. Sodium which is principle cation of extracellular fluid, contributes major portion of to the osmotic pressure (90%).

Since the cell membranes are completely permeable to water, the effective osmotic pressure in the two compartments are considered to be equal. Any condition that alters the effective osmotic pressure in the two compartment will result in redistribution of water between the compartments. Thus an increase in effective osmotic pressure in the extracellular fluid, which would occur most frequently as a result of increased sodium concentration will cause a net transfer of water and would continue until the effective osmotic pressure in two compartments are equal. Thus intracellular fluid, shares in losses that involve a change in concentration or composition of

the extracellular fluid but shares slowly in changes involving loss of isotonic volume alone. Serum osmolality, normally 289±9 Osmol/kg measures the total concentration of all osmotically active entities in serum water, like other measurements of concentration its does not of itself give information about the total amounts of circulating materials which depends on the plasma volume as well as the concentration. Additional information is obtained if urinary osmolality is measured simultaneously as this reflects the action of anti-diuretic hormone on renal tubules.

Increase in serum osmolality is a consequence of either an increase in serum sodium concentration or in the concentration of other osmotically active substances.

The decrease in serum osmolality is almost always attributable to a low sodium concentration. Although this may be due to sodium deficiency the more marked falls are seen in condition with water retention without sodium retention. Excess ADH activity which is a feature of body's response to injury may also lead to low serum osmolality particularly if excessive intravenous administration of Isotonic glucose solution is carried out after trauma or in the immediate post operative period.

Osmolar gap :- The difference between the measured plasma osmolality & osmolality predicted from measured (Na^+) is termed as osmolar gap.

EFFECT OF ANAESTHESIA ON SODIUM BALANCE

Fall in serum sodium level is well known to occur after any surgery (Flear et al, 1980 and Chan et al, 1980). Changes in fluid and electrolyte metabolism as a result of trauma are for the most time results of alternation in systemic neuroendocrinal enviorment. In order for a reflex to be initiated, the stimulus must be perceived by a specialized receptor that transduces the stimulus in to electrical activity and trasmits to brain. This exemplified by the experiments of Lune & Egdah (1959) inwhich one hindlimb of dog was left attached to body only by femoral nerve, artery and vein. The trauma to the innervated but otherwise detached limb continued to evoke an increased A.C.T.H. & cortisol response. When nerve was severed, leaving only the artery & vein intact the response to trauma was eleminated. Similarly patients undergoing lower limb surgery under spinal anaesthesia donot demonstrate an increase in vasopressin sedretion during the procedure as compared to patients under going the same procedure under general anaesthesia. This is because of inhibiting effect of spinal anaesthesia on neural pathways.

Laprotomies in the absence of diminished circulating volume do not result in adrenocortical stimulation if the traumatized area is dennervated. Similarly local anaesthetics by blocking the transmission of afferent impulses from the area of injury, inhibit the neuroendocrinal response to operative trauma elicited by stimulus present at the operative site. The perception of stimulus need not be concious, as evidenced by the ability of individuals to respond to surgical stimulus despite the presence of general anaesthesia. Even this response is not the same had anaesthesia not been present. The difference arises at least in part, through ability of general anaesthetics themself to initiate, inhibit or augument neuroendocrinal reflexes.

No operative trauma ought to be thought of without consideration of the particular anaesthetic agent employed & depth & duration of anaesthesia.

The sodium response of surgical trauma

Any surgical trauma causes a sudden rise in aldosterone & cortisol level in the patient (Liaurado J.C. & N.F.Woodruff 1957). The aldosterone & cortisol, both are responsible for sodium retention in the post operative periods (Japson R.P. K.M. Endoer

1951). The decreased renal excretion of sodium is a well documented feature of post operative periods (Hardey J.D. & I.S. Ravdin 1952). Functional intracellular fluid volume has recently been shown to be a major determinant of the magnitude of renal sodium excretion in normal individuals (Epstein F.H. 1957). The reduction of functional extracellular fluid volume during the operative procedure is independent of whole blood loss during operation. The only factor observed which would tend to influence the degree of functional extracellular loss is the magnitude of the local trauma. Thus a decrease in functional extracellular fluid volume during post operative period is due to an internal redistribution of fluids. This decreases in functional intracellular volume in itself is a strong stimulus for aldosterone secretion despite on over all fluid retention. This reduction of ECF volume in turn is responsible for sodium retention in the post operative periods. Thus normally there is sodium retention in post operative periods.

A fall in plasma sodium concentration often to hyponatremic level, is well known to occur after trauma and major surgery (Flear C.T.G. Bhattacharya S.S., Singh C.M., 1871 Chan S. Redcliffe & A. Johnson 1980). It is widely believed that fall

in plasma sodium after uncomplicated surgery results from endogenous dilution, but this is insufficient to cause all of the observed changes. In patients severely ill after operation profound fall in plasma sodium may occur and osmolar gaps seen (Tindell S.F., Clark R.G. 1976 & Flear C.T.G., Singh C.M. 1963). The lowering of plasma sodium may be abrupt or slow & sustained. Abrupt fall in sodium plasma are often accompanied by reduced osmolality. The sick cell concept attributes osmolar gaps to isotonic redistribution of solutes, from cells to extracellular fluid, caused by an abrupt increase is cell membrane permeability and the sustained dilution with no osmolar gaps to a wide spread impaired capability of cells to maintain their normal content of non diffusable solute (Flear C.T.G., 1970, Flear C.T.G. Singh C.M., 1972, 1973).

The fall in serum sodium has been seen in the presence of sodium retention after trauma, inspite of a raised aldosterone level. Part of this hyponatremia can be explainable on the basis of obligatory antidiuresis due to raised antidiuretic harmone level lasting for 24-36 hours (LeQueane & Lewis 1952) post operatively. The plasma aldosterone concentration demonstrates a circadian rhythum in which the peak concentration is late afternoon and night. Following trauma, surgery, the circadian rhytum is lost 'elevated concentration are observed during entire 24 hours

period. Plasma concentration of aldosterone also increases following anaesthesia alone, but not to the extent seen, following injury ' major operations. The highest concentration of aldosterone has been noted in the agonal period following injury.

Cells of adrenal cortex, zone glomerulosa synthesize and secrete aldosterone in response to trauma, The two most important mechanism for aldosterone secretion appear to be through A.C.T.H. & angiotensin. Stress induced elevation in aldosterone is probably mediated through A.C.T.H. The stimulatory effect of A.C.T.H. on aldosterone production is short lived. As a result of this short lived potency. A.C.T.H. probably has a minor role in chronic status where angiotension II appears to be the main stimulating hormone. Other factors that may alter the aldosterone secretion by adrenal cortex

1. Increased P_k (Plasma potassium).
2. Decreased P_{Na} (Plasma sodium)

Increase in P_k represent an important stimuli for aldosterone secretion, but does not represent a mechanism for changing aldosterone secretion when sodium intake changes. The increased aldosterone secretion seen with decreased plasma sodium represents an appropriate response for maintaining the sodium balance. However the effect of plasma sodium on aldosterone secretion is of minor importance in the regulation of sodium excretion for two reasons,

first of all, decrease in plasma sodium has a relatively weak stimulatory effect on aldosterone secretion, secondary changes in sodium intake have minimal effects on plasma sodium. For example, while an increase in sodium intake adds sodium to the extracellular fluid & produces a transient increase in plasma sodium, The plasma osmolality also increases, stimulating osmoreceptors. The resulting stimulation of thirst & ADH release leads to expansion of the plasma volume and dilution of the ingested sodium, so that the over all changes in plasma sodium is small. Thus the changes in aldosterone secretion that accompany changes in sodium intake must be primarily mediated by Angiotensin II.

Primary action of aldosterone is related to fluid & electrolyte balance. In the early distal convoluted tubules aldosterone increases the reabsorption of sodium & of chloride & in late convoluted tubules & early collecting duct it promotes reabsorption of sodium & excretions of potassium. Thus aldosterone level during & after surgery (trauma), is responsible for the sodium retention, but hyponatremia in post operative period is provoked by an even greater gain of water (C.M.Singh & C.T.G. Flear 1968) due to persistent elevated level of antidiuretic hormone in serum. In experimental animals when the factors that regulate sodium excretion like G.F.R. & aldosterone,

are controlled an animal can still regulate sodium excretion to match sodium input. For example, in an experimental animal if constant G.F.R. is maintained by controlling blood flow to the kidneys & high plasma concentration of aldosterone is maintained by administerating large doses of hormone, intravenous infusion of isotoinic saline still will be followed by a decrease in sodium reabsorption & hence an increase in sodium excretion. The phenomenon whereby an increase in sodium input can result in an increase in sodium excretion independent of any significant increase in G.F.R. or decrease in aldosterone level is termed as " Third factor effects". Conversely a decrease in sodium input can result in decrease in sodium excretion independent of any significant changes in G.F.R. or aldosterone level a phenomenon that can be referred to as the absence of third factor.

The mechanism for third factor effect remains poorly understood (Dewardner 1978). The effects probably involves various mechanisms because changes in sodium reabsorption in both proximal tubules & distal nephron are observed. With small increase in sodium intake, third factor effect appears primarily due to decrease in sodium reabsorption in medullary collecting ducts. It has been postulated that this decrease in sodium reabsorption is mediated by prostaglandins, bradykinon or as an yet unidenti-

fied nature uretic hormone when large quantities of sodium are administered (intravenous infusion) sodium reabsorption in P.C.T. is depressed correspondingly. Thus decrease in proximal tubular sodium reabsorption can be attributed to dependence of proximal tubular water & solute reabsorption on the hydrostatic & oncotic pressure in the peritubular cappilaries. According to starling's principle, " Role of fluid movement from capillaries to interstitial space is proportional to the difference between hydrostatic & oncotic pressure gradients across the capillary wall, the so called net filteration pressure.

Rate of filteration is directly porportional to $(P_c - P_1) - (\bar{\pi}_c - \bar{\pi}_1)$. The rate of fluid movement from interstitial space to capillaries (rate of reabsorption) is therefore proportional to $(P_1 - P_c) - (\bar{\pi}_1 - \bar{\pi}_c)$.

If above equation is applied to reabsorption of fluid from peritubular intertitial space into peritubular capillaries, it becomes evident that an increase in peritubular capillary hydrostatic pressure (P_c) or decrease in peritubular capillary oncotic pressure $(\bar{\pi}_c)$ will retard reabsorption of fluid into capillaries. The movement of fluid from lateral intracellular space to the peritubular space will therefore be retarded & hydrostatic pressure in lateral space would increase. This increased hydrostatic pressure in turn will impair reabsorption of

water & solute by P.C.T. Ingestion of large quantity of sodium increase peritubular capillary hydrostatic pressure & decreases peritubular oncotic pressure, there by decreasing reabsorption of sodium by proximal tubular, such change in peritubular capilla-ry & hydrostatic & oncotic pressure also would decrease reabsorption of water & other solutes by proximal tubules. This shows that all P.C.T. reabsorption is decreased following ingestion of large quantities of sodium or as a result of plasma volume expension, Many investigators believe that a hormone also may be involved. It is not known whether this natriuretic hormone is same as hormone postulated to decrease sodium reabsorption in medullary collecting duct in response to small increase in sodium. The detailed mechanism for third factor effect are not completely understood, the participation of both proximal tubule & medullary collecting ducts occur in logical manner with small increase in sodium intake, the third factor effect occurs in the region of the nephron that re-absorbs small quantities of sodium & "five times the rate of sodium excretion i.e. the medullary collecting ducts. With large increase in sodium intake, third factor effects also occurs in region that absorbs large quantity of sodium i.e. P.C.T.

The other hormone playing an important role in sodium & water regulation is arginine vasopressin (antidiuretic hormone) which is the primary hormone of neurohypophysis in human beings. This hormone is synthesized in hypothalmus & transported to posterior pituitary, where it is stored & secreted when a stumulus comes to the posterior pituitary. There are many stimuli to activate posterior pituitary & one of them is any surgical process or trauma.

Moran et al (1964) have identified four phases of vasopressin secretion following surgery. The first phase is normal preoperated control period in which plasma vassopressin concentration is within normal range. The second phase consists of mild elevation that results from overnight fast. This period can be abolished by administration of I/V fluids during the preoperative period. The third phase results from cutaneous & visceral stimuli & lasts from skin incision to closure. This phase is characterized by transient elevation of antidiuretic hormone. The fourth phase corresponds to the post operative phase in which there is an early increase is the plasma vassopressin concentration followed by a return to normal value by fifth post operative day.

Moran et al (1964) also hypothetized that there are four efferrent reflaxes controlling vasopressin release & each of these can over ride the preceeding one three of these reflaxes osmoreceptor, Baroreceptor, & left atrial stretch receptor reflax are negative feed back loop. Therefore in the presence of pain vasopressin secretion can occur in the face of a hypoosmolar hypovolomic condition that would normally inhibit vasopressin secretion & may explain persistent elevation of vasopressin secretion seen for 5-7 days following surgery. The persistent secretion of vasopressin produces a low urinary output with high osmolality & profound dilutional hyponatremia.

Any surgery, produces rapid changes in functional extracellular fluid volume (Shires et al 1961), effective circulatory volume, extracellular osmolality & electrolyte composition, that results in the stimulation of the neuroendocrine system. Thus neuroendocrinal response induces alteration in the renal and circulating functions which can then alter the salt & water balance as required.

The increase in plasma vasopressin lasts for 3-5 days after surgery under & in most circumstances it results in water retention & oliguria.

Post operative oliguria was originally beleived to be a normal accompaniment of surgery & did not have ill effects.

Shires et al (1961) hypothetized that a significant loss of fluid in 3rd space may account for part of hyponatremia. The osmolar gaps seen in most operative patients could have been due to an isoosmolar redistribution of solute caused by an increased cell membrane permeability (Flear P, Singh 1983, 1978, 1982).

Role of fluid administration in surgical patients:-

Infusion of saline less fluid plays an important part in post operative hyponatremia (Chan et al 1980). Thus serum sodium level can be affected by the type of fluid given in two ways.

1. Predisposes to acute tubular necrosis in patients with severe trauma in which hypovolamia & hypotension tends to occur.

2. It sets the stage for development of water intoxication (severe dilutional hyponatremia, if large volumes of solute free fluids are given to patients before during or immediately after operative events. Thus most common electrolyte abnormality seen following surgery namely hyponatremia, is partly as a result administration of hypotonic fluid even under conditions that favours salt & water retention.

The action of vasopressin in effecting water retention requires the presence of an intact counter current mechanisms in the loop of henle. This counter current mechanism is disrupted by a fall in medullary osmolality, since maintainence of normal medullary osmotic gradient requires the adequate delivery of sodium & chloride, to the long loops of Henle, which is decreased frequently after injury. The action of vasopressin is then impaired resulting in a defect in the urinary concentration motility. Thus abnormal or increased urinary output in a hypotensive or surgical patients does not reflect an adequate blood volume. In order to combat the fall in medullary gradient following surgery,adequate tubular fluid flow must be ensured & maximal sodium reabsorption in proximal nephron must be avoided. This is accomplished by administration of liberal amounts of salt solution such as ringer lactate or normal saline in the early post operative periods.

The administration of normal saline in the early post operative periods may result in a marked positive sodium & solute balance which may cause edema. During this period of increased vasopressin secretion the urine volume can not be increased by administration of water alone. It is the

solute lead that determines urine volume & free water clearance during this period. An increase of urine output will occur only after extracellular fluid space has been expanded by increasing solute load. This increased urine output may result in puffy patient postoperatively, but minimizes the protection of renal function.

That dilution alone is insufficent to account for the fall observed in the serum sodium has been shown by the relationship between serum osmolality & the serum sodium (Flear et al 1980, Singh & Singh, 1971), in the peroperative & immediate post operative period. Among the two phases of water retention after surgery described by LeQueane & Lewis (1953), the initial obligatory antidiuretic phase occured irrespective of sodium content of fluid infused and was not suppresed by a strong hypotonic stimulus of dilutional hyponatremia, Which may occur if the patient is given only dextrose solution or by the isotonic expansion of extracellular space if patient is given isotonic solution. This could be because the water retention which is taking place, is controlled by vasopressin (Thomas & Morgan 1979 & Sinnetamby et al 1974) and sodium retention by aldo-

sterone and other factors (Coeheran 1978). The second phase of fluid retention which lasts from 36-129 hours is effected by the sodium content of infused fluid. Therefore administration of sodium free fluids leads to hyponatremia with diureases with free water loss (Tindell & Clark 1981). This may be either because of the sodium wash out effect in diuresis or the resetting of osmoreceptor in hypothalmus on a lower level (Robertson & other 1976).

The patients who had been given only saline on the other and tend to retain and maintain plasma sodium level. The reason for sodium retention and normal serum sodium level is difficult to understand. There are several explanations for overriding of the vasopressin response. the presence of isotonic expansion of extracellular space may have allowed stretch receptors in the capacitance vessels to readjust without incereasing the vasopressin secretion. Alternatively the kidney may have developed a reduced ability to excrete sodium in the late post operative period & this could have led to sodium retention in the presence of a high sodium intake (Fuidal et al 1981). In post operative period there is mild to moderate hyponatremia with hyperkalaemia, this is primarily brought about by the secretion of vasopressin, plus the overhydration of

patients with non solute containing fluid. The potassium level may be some what elevated. Potassium is lost from cells as a consequence of surgical trauma, corticosteroid level & starvation.

Patients after surgery have a diminished urinary excretion of sodium & they do not excrete all the sodium load, which is attributed to reduced plasma volume, Flear & Clark (1953) observed that sodium retention did not occur after trauma if the patient were given adequate blood transfusion or isotonic solution. Irvin et al (1972) reported that the urine sodium did not fall in patients after surgery if they were given balance salt solution during as well as after the surgery.

The fall in plasma sodium which occurs in the majority of patients given saline or dextrose saline after surgery is much swollen than seen that in patients with symptomatic water intoxication after surgery (Dontsen et al 1966).

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MATERIAL AND METHOD

MATERIAL AND METHODS

The study of serum electrolytes was done on three groups of patients, 15 to 75 years under going surgery and requiring fluid infusion for at least 24 hours post operatively.

The patients were divided into three groups according to nature of fluid infused in post operative period.

Group I : 3 lit. of Dextrose 5% /day

Group II : 1 lit. of Isotonic saline }
2 lit. of Dextrose 5% } /day

Group III : 2 lit. of Isotonic saline }
1 litl of Dextrose 5% } /day

Investigation

1. Blood pressure - Lying down
   - Sitting posture
2. Haematocrit
3. Serum studies - S.Sodium
   - S.Potassium
   - S.Osmolality
   - Blood sugar level
   - Blood urea level
4. Urine analysis - Volume / 24 hours
   - Urine sodium excretion
   - Specific gravity
5. Body weight

On the day before operation patients were starved from midnight except in emergency surgery. The intravenous fluid was administered through peripheral veins. All the patients were either operated under general anaesthesia or in spinal anaesthesia. The analgesic given during per operative or post operative period was similar. The blood sample taken at 0900 hour from peripheral vein on day 1 (one day prior to operation, '0' immediate post operative day, + 1 (first post operative day), + 2 (second post operative day) and subsequently, by standard technique with a sterilized syringe and needle.

24 hours urine was collected or patient was catheterized to measure exact 24 hours urine volume.

The serum sodium, potassium and urinary sodium were measured by flame photometer in the department of Biochemistry, M.L.B. Medical College, Jhansi.

Principle- When small quantity of metal salts such as sodium or potassium is introduced into a flame, a characterstic light is emitted. The measurement of the intensity of such emission and its correlation with the concentration of the element is the basis of flame photometery.

The instrument made on this principle is called flame photometer. The flame photometer has the following parts.

1. Air pressure regulator and flow meter for the fuel gas.
2. Atomizer
3. Burner
4. Optical system
5. Photocells
6. Recorder - A galvanometer with light spot or needle for the fuel gas, cooking gas cylinder is convenient. Compressed air is used to atomize the sample and carry it to non luminous flame. Both gas and air supplies are carefully regulated to maintain constant flow rate of the samples into the flame.

The solution is sprayed as a fine mist of droplets in the non luminous flame which becomes coloured by the characteristic emission of the metal light of wave length which corresponds to the element being determined is isolated by the use of light filter or prism system and allow to fall on photocells. The electric current generated is measured. This is indicated by the light spot on the recorder.

Determination of serum sodium & potassium & urinary sodium

Material and reagents

1. Flame photometer
2. Gas cylinder
3. Polythane & bottles (500 mg) for standard solution
4. Polythane container for distilled water
5. Polythane small cuvottes for aspirating test solution in flame.
6. Double distilled or deconized water
7. Polythane small tubes
8. Stock sodium standard (200 meq/lt.) dissolve 11.69 gm of pure dry sodium chloride (Nacl) in one lit. of water.
9. Stock potassium standard (10 meq/lt.) dissolve .746 gm of pure dry potassium chloride (Kcl) in one lit. of water.
10. Combined working standard of sodium & potassium

Procedure

Sodium - Put the light filter (580 - 590 non yellow green) in the filter socket, adjust the gas adjusting pnob gradually untill individual blue comes of flame become separated. Then adjustment of glavanometer is done. First with distilled water and than with minimum strength working solution. Then aspirate one by one standard solution and note the glavanometer reading and than calculated sodium levels.

<u>Potassium</u> - The potassium light filter (766-700 mm Red) The instrument is standardized and the same test solution is aspirated and reading is noted and potassium value calculated.

<u>Urinary sodium</u> - Dilute the urine 1 to 100 ml and measurement is done as for blood sodium.

It is sometimes helpful to calculate osmolality from the molar concentration of main osmotically active substances. For both serum & urine this can be done if molar concentration of so (Na^+), K^+ urea and glucose and known. The serum osmolality is calculated by formula (Harrison's principle of internal medicine II D.N. 1791).

S.Osmolality (osmol/L)

$$= 2(Na^+) + (K^+) + \frac{\text{Glucose(mg/100ml)}}{18} + \frac{\text{Urea(mg/100ml)}}{2.8}$$

For most normal sera this is close to $2(Na^+ + K^+)$ and for normal urine glucose can be ignored comparison of calculated osmolality with that actually determined is often helpful in painting to the presence of some previously unsuspected osmotically active substance. The specific gravity of urine was measured by urometer.

The weight of patient was recorded weight 0200 hours such day after correcting it for the weight loss resulting from the removal of surgical specimens.

O B S E R V A T I O N

OBSERVATIONS

The present study was done in our institute, M.L.B. Medical College & Hospital, Jhansi between April, 1990 to May, 1991. During the above mentioned period, we studied the effect of type of fluid infusion during perioperative period on serum electrolytes, in patient undergoing surgical procedures. During this period 126 patients were studied for changes in serum electrolytes' values in relation to type of fluid infusion.

The fluid was started at the time of operation and continued till needed. During pre operative and subsequent post operative days 3 (lt.) of fluid was given intravenously/day.

The type of fluid infusion was divided into three groups.

<u>Groups</u>

1. Patient receiving 5% dextrose 3 (lit.)/day.
2. Patient receiving 5% dextrose 2 (lit.) + Isotonic saline 1 lit./day
3. Patient receiving 5% dextrose 1 lit. + Isotonic saline 2 lit./day

All 126 patient were studied for

- Serum sodium

- Serum potassium
- Blood sugar
- Blood urea
- Urine volume
- Specific gravity of urine
- Body weight
- Serum osmolality

The various values were tabulated, and subsequent calculation of mean value, standard deviation & test of significance between two means (P value) was done.

Serum osmolality was calculated as:

1. Serum osmolality (osmol/lit.)

$$= 2(Na^+) + (K^+) + \frac{\text{Blood Glucose mg/100ml}}{18} + \frac{\text{BUN mg/100ml}}{2.8}$$

2. Mean (X) $= \dfrac{\text{Total value}}{\text{Total number of patient}}$

3. S.D. $= \sqrt{\dfrac{(X - X)\ 2}{n}}$

4. P(Test of significance)

$$t = \frac{\overline{X}_1 \quad \overline{X}_2}{s \sqrt{\frac{1}{N_1} + \frac{1}{N_2}}}$$

1. <u>Sex distribution (Table No. I)</u>

   Out of 126 patient studied 111 were males & 15 females. or

   88.09% and 11.91% respectively.

2. Fluid distribution (Table No. II)

Out of 126 patient studied

Group I - 45 patients were given 5% dextrose 3 lit.
per day, invusion I/V pre & postoperatively.

Group II- 48 patients were given 5% dextrose 2 lit.
+ Isotonic saline 1 lit./day.

Group III-33 patients were given 5% dextrose 1 lit.
+ Isotonic saline 2 lit./day.

3. Serum sodium (table III)

Serum sodium level variation, in various groups can be seen, from table III.

In group I, patient receiving 5% dextrose 3 lit. intravenously there was a significant fall of serum sodium level (P value) $\angle$ .001 on first post operative day & serum sodium fall continued in IInd & IIIrd post operative day (P value) $\angle$.005.

In group II patients receiving 5% dextrose 2 lit. + Isotonic saline 1 lit./day there was a significant fall on first post operative day & subsequent post operative days while in IIIrd group i.e. patient receiving 5% dextrose 1 lit. + Isotonic saline 2 lit. there is a insignificant rise of serum sodium (P value) $\angle$.5.

<u>Serum Potassium (Table IV)</u>

Patient receiving 3 lit. of 5% dextrose /day showed a rise of ($P < .005$) on first post operative day and which persisted during the subsequent post operative days.

Patient receiving 2 lit. of dextrose 5% + 1 lit. of isotonic saline /day in did not show any significant rise in serum potassium.

Patient receiving 1 lit. of 5% dextrose + 2 lit. isotonic saline/day showed a significant rise ($P < .05$) on first post operative day and which persisted on subsequent post operative days.

<u>Serum Osmolality (Table V)</u>

- Patient receiving 3 lit. of 5% dextrose/day showed significant fall in mean value of serum osmolality (P value $< .005$) on first post operative day and it persisted on second, third and fourth post operative day.
- Patient receiving 2 lit. of 5% dextrose + 1 lit. of isotonic saline/day showed significant fall in serum osmolality (P value $< .05$) on first and second post operative day.
- Patient receiving 1 lit. of 5% dextrose + 2 lit. isotonic saline had an insignificant rise in($P < .5$) in serum osmolality on first & subsequent post operative days.

Blood sugar and blood urea (Table VIII)

All the three groups of patient receiving 3 lit. of 5% dextrose, 2 lit. of 5% dextrose + 1 lit. of Isotonic saline, 1 lit. of 5% dextrose + 2 lit. of isotonic saline, showed an insignificant fall in blood sugar level while there is no change or an insignificant rise of blood urea level post operatively.

Weight (Table VI)

It was difficult to weight patients on first post operative day. But on second & third post operative day there was a insignificant fall in weight in all three groups of patient receiving 3 lit. of 5% dextrose 2 lit. of 5% dextrose + 1 lit. isotonic saline & 1 lit. of 5% dextrose + 2 lit. of isotonic saline.

Urine output (Table VII)

Patients receiving 3 lit. of 5% dextrose /day & 2 lit of 5% dextrose + 1 lit of isotonic saline showed a significant rise ($P\angle.001$) on first post operative day which persisted on second and third post operative days while in third group i.e. patient receiving 1 lit. of 5% dextrose + 2 lit. isotonic saline there was a insignificant rise in urine output ($P\angle.05$) on first post operative day and significant

rise in ($P < .001$) urine output an second & third post operative day. Urine output is increased in each group upto second day and than it become constant.

Specific gravity - There was an insignificant change in specific gravity of urine of all patients.

Urine sodium

Patient receiving 3 lit. of 5% dextrose/ day showed a significant fall in urine sodium level ($P < .005$) on first post operative day but on second post operative day onwards urine sodium changes were insignificant ($P < .5$).

In patient receiving 2 lit. of 5% dextrose + 1 lit. of isotonic saline there is significant fall in urine sodium level in post operative period.

In patient receiving 1 lit. of 5% dextrose + 2 lit. isotonic saline there is an insignificant fall in urine sodium level ($P < .5$) on first post operative day but significant rise of urine sodium from second post operative day & onwards.

Incidence of hyponatremia (Table IX)

(Serum sodium level below 135 mmol/lit.) In various groups receiving different type of fluid showed variation in serum sodium level.

In group I - patient receiving 3 lit. of 5% dextrose, 20% of patient are pre-operatively hyponatremic. After 3 lit. of 5% dextrose infusion per day percentage of patient becomes hyponatremic rises to 60% in first post operative day and 73% to second post operative day. In group IIrd 12.5% patient are hyponatremic pre operatively which increases to 37.5% in first post operative day after receiving 2 lit. of 5% dextrose + 1 lit. of isotonic saline/day while percentage of patient becomes hyponatremic, increases to 43.75% on the second post operative day.

In groups IIIrd receiving 1 lit. of 5% dextrose + 2 lit. of isotonic saline 9% of patients are hyponatremic pre operatively and same percentage of patients remain hyponatremic post operatively.

TABLE NO. 1

Sex distribution of patient undergoing surgical procedure with variation in fluid infusion.

Sl. No.	Number of patient		Percentage
	Sex	Numbers	
1.	Male	111	88.09%
2.	Female	15	11.91%
Total		126	

TABLE NO. II

Fluid distribution of patient under going surgical procedure.

Sl. No.	Type of fluid injured to patient in 24 hours	Number of patient
1.	Dextrose 5% (3 lit.)	45
2.	Dextrose 5% (2 lit.) + Isotonic saline (1 lit.)	48
3.	Dextrose 5% (1 lit.) + Isotonic saline (2 lit.)	33
Total		126

Mean values of serum sodium & urinary sodium in patients undergoing surgical procedure.

Sl. No.	Nature of fluid infused /24 hrs.	Number of patient	Values	SERUM SODIUM					URINARY SODIUM				
				Pr.O.	Po_1.O	Po_2.O	Po_3.O	Po_4.O	Pr.O	Po_1.O	Po_2.O	Po_3.O	Po_4.O
1.	Dextrose 5% 3 lit.	45	Mean	141.19	135.71	132	131.5	135	86.7	67.68	86.82	91.2	55
			S.D.	± 6	± 5.06	±4.95	±3.11	± .7	± 48.92	±29.02	±35.26	±33.24	±17
			P	-	< .001	< .005	< .005	< .001	-	< .005	< .05	< .05	-
2.	Dextrose 5% 2 lit. + Isotonic saline 1 lit.	48	Mean	139.51	138.51	137.56	139.5	130.5	97.02	86.62	83.73	77.18	89.5
			S.D.	±6.46	± 7.10	±5.71	± 7.13	± 6.5	±38.5	±34.2	±39.33	±39.84	±62.5
			P	-	< .05	< .01	< .05	< .001	-	< .05	< .05	< .005	< .5
3.	Dextrose 5% 1 lit. + Isotonic saline 2 lit.	33	Mean	140.83	142.38	141.18	140.31	-	91.35	87.74	132.2	144.72	-
			S.D.	± 3.84	± 5.2	± 4.95	±5.64	-	±28.64	±30.86	±27.95	±24.98	-
			P	-	< .5	< .5	< .5	-	-	< .5	< .5	< .5	-

S.D. = Standard Deviation
P = Test of significance
Pr.O = Pre operative
Po.O = Post operative

Po_1 = First post operative day
Po_2 = Second Post operative day
Po_3 = Third Post operative day
Po_4 = Fourth Post operative day

TABLE NO. IV

Mean values of serum potassium in patient undergoing surgical procedure.

Sl. No.	Type of fluid infused /24 hours	Number of patients	Values	Pr.O	Po_1.O	Po_2.O	Po_3.O	Po_4.O
1.	Dextrose 5% 3 lit.	45	Mean	4.24	4.47	4.08	3.81	4
			S.D.	±.47	±.76	±.37	±.68	±.4
			P	-	$<$ .05	$<$ .01	$<$ .05	$<$ .5
2.	Dextrose 5% 2 lit. + Isotonic saline 1 lit.	48	Mean	4.16	4.19	4.23	4.12	4.38
			S.D.	±.74	±.39	±.46	±.42	±.4
			P	-	$<$ .5	$<$ .5	$<$ .5	$<$ .5
3.	Dextrose 5% 1 lit. + Isotonic saline 2 lit.	33	Mean	3.51	3.87	3.92	3.92	-
			S.D.	±.51	±.41	±.3	±.29	-
			P	-	$<$ .05	$<$ .005	$<$ .005	-

TABLE NO.V

Mean value of serum osmolality of patient undergoing surgical procedure.

Sl.No.	Type of fluid infused/24 hrs.	Number of patient	values	Pr.O.	$Po_1.O$	$Po_2.O$	$Po_3.O$	$Po_4.O$
1.	Dextrose 5% 3 lit.	45	Mean	299.96	289.42	277.68	280.79	281.85
			S.D.	±11.76	±9.65	±9.76	± 7.27	±10.51
			P	-	<.005	<.005	<.005	<.005
2.	Dextrose 5% 2 lit. + Isotonic saline 1 lit.	48	Mean	297.22	294.09	292.5	295.09	282.18
			S.D.	±12.66	±13.36	±11.28	±14.89	±12
			P	-	<.05	<.05	<.5	<.05
3.	Dextrose 5% 1 lit. + Isotonic saline 2 lit.	33	Mean	297.73	298.92	296.4	293.58	-
			S.D.	±7.26	±8.55	±9.05	±9.26	-
			P	-	<.5	<.5	<.05	-

TABLE NO. VI

Mean value of weight of patient undergoing surgical procedure

Sl. No.	Type of fluid infused/24 hours.	Number of patient	values	Pr.0	Po_1.0	Po_2.0	Po_3.0	Po_4.0
1.	Dextrose 5% 3 lit.	45	Mean	54.33	-	53.46	56.85	58
			S.D.	± 9.39	-	± 9.18	± 6.07	± 8
			P	-	-	$<.5$	$<.5$	$<.5$
2.	Dextrose 5% 2 lit. + Isotonic saline 1 lit.	48	Mean	55.96	-	55.8	52.41	43
			S.D.	± 8.01	-	± 8.21	± 9.22	± 7
			P	-	-	$<.5$	$<.5$	$<.5$
3.	Dextrose 5% 1 lit. + Isotonic saline 2 lit.	33	Mean	56.90	-	56.95	57.7	-
			S.D.	± 4.42	-	± 4.7	± 4.87	-
			P	-	-	$<.5$	$<.5$	-

TABLE NO. VII

Mean values of specific gravity of urine & urine volume /day of patient undergoing surgical procedure.

Sl. No.	Type of fluid infused /24 hrs.	Number of patient	Values	Specific gravity					Urine volume				
				Pr.O	Po_1.O	Po_2.O	Po_3.O	Po_4.O	Pr.O	Po_1.O	Po_2.O	Po_3.O	Po_4.O
1.	Dextrose 5% 3 lit.	45	Mean	1.015	1.009	1.014	1.018	1.011	1550	2050	2200	1900	2250
			S.D.	±.007614	±.6081	±.0092	±.0082	±.6082	±425	±604	±450	±399	±350
			P	-	∠.5	∠.5	∠.5	∠.5	-	∠.001	∠.001	∠.001	∠.001
2.	Dextrose 5% 2 lit. + Isotonic saline 1 lit.	48	Mean	1.018	1.012	1.014	1.021	1.016	1000	1500	1800	1750	1600
			S.D.	±.0082	±.0072	±.0083	±.0092	±.028	±302	±489	±580	±438	±400
			P	-	∠.5	∠.5	∠.5	∠.5	-	∠.001	∠.001	∠.001	∠.001
3.	Dextrose 5% 1 lit. + Isotonic saline 2 lit.	33	Mean	1.018	1.014	1.010	1.010	-	1100	1200	1600	1800	-
			S.D.	±.0082	±.0086	±.0092	±.0078	-	±106	±690	±384	±340	-
			P	-	∠.5	∠.5	∠.5	-	-	∠.5	∠.001	∠.001	-

TABLE NO. VIII

Mean values of blood sugar & blood urea in patients undergoing surgical procedure

Sl. No.	Type of fluid infused /24 hours.	Number of patients	Values	Blood Sugar					Blood Urea				
				Pr.0	$Po_1.0$	$Po_2.0$	$Po_3.0$	$Po_4.0$	Pr.0	$Po_1.0$	$Po_2.0$	$Po_3.0$	$Po_4.0$
1.	Dextrose 5% 3 lit.	45	Mean	98.86	97.86	98	95.6	93	29.53	29.2	27.73	27.1	26
			S.D.	±11.4	±18	±17.13	±18.42	± 7	±9.28	±6.87	±5.94	±6.48	± 2
			P	-	∠.5	∠.5	∠.5	∠.5	-	∠.5	∠.5	∠.5	∠.5
2.	Dextrose 5% 2 lit.+ Isotonic saline 1 lit.	48	Mean	94.75	95	98	91.81	111	27.25	27	25.62	24.92	29.5
			S.D.	±21.94	±22	±19.82	±19	± 13	±7.87	±7.83	±8.1	±7.98	±2.5
			P	-	∠.5	∠.5	∠.5	∠.5	-	∠.5	∠.5	∠.5	∠.5
3.	Dextrose 5% 1 lit. + Isotonic saline 2 lit.	33	Mean	95.49	94.36	93.81	93.5	-	22.9	23.18	21.09	19.6	-
			S.D.	±10.71	±9.33	±9.38	±10.62	-	±8.58	±8.94	±5.09	±4.04	-
			P	-	∠.5	∠.5	∠.5	-	-	∠.5	∠.5	∠.5	-

TABLE NO. IX

Number of patient showing hyponatremic (Serum sodium level post operatively ∠ 135 mmol/Lt.).

Sl. No.	Type of fluid infused /24 hrs.	Number of patient	Pr.O	Po_1.O	Po_2.O	Po_3.O	Po_4.O
1.	Dextrose 5% 3 lit.	45	9(20%)	27(60%)	33(73.33%)	27(60%)	-
2.	Dextrose 5% 2 lit. + Isotonic saline 1 lit.	48	6(12.5%)	18(37.5%)	21(43.75%)	12(25%)	-
3.	Dextrose 5% 1 lit. + Isotonic saline 2 lit.	33	3(9%)	3(9%)	3(9%)	3(9%)	-

D I S C U S S I O N

DISCUSSION

Serum sodium in relation to surgical procedure and fluid administration.

Serum sodium constitutes the main cation in extracellular fluid which maintains the osmolality of extracellular fluid. The serum sodium level normally represents the degree of dilution or concentration of extracellular body fluid both in health and disease. Retention of sodium, reduction in urinary sodium and reduction of free water excretion are the classic responses to surgical trauma. The surgical trauma causes a sudden rise in aldosterone & cortisol level. The cortisol & aldosterone both are responsible for sodium retention and reduction in urinary sodium in post operative period (Japson R.P., 1956; K.M.Chadon 1951).

Following surgery, important mechanism for aldosterone secretion appears to be through A.C.T.H.. The stimulatory effect of A.C.T.H. on aldosterone production is short lived. As a result of this short lived potency A.C.T.H. probably has a minor role in chronic states where angiotension II appears to be the main stimulatory hormone, which in addition also has a stimulatory role even in the early phase of injury. Other factors that may alter the aldosterone secretion by adrenal cortex are

1. Increased pK^+ (Plasma potassium)

2. Decreased pNa^+ (Plasma sodium)

Increased potassium level is a good stimulus for aldosterone secretion but does not regulate the sodium level, while increased aldosterone seen with decreased plasma sodium represents on appropriate response for maintaining sodium balance. However the effect of plasma sodium on aldosterone secretion is of minor importance in regulation of sodium excretion.

1. Decrease in plasma sodium has a relatively weak stimulatory effect on aldosterone secretion.
2. Changes in sodium intake have minimal effect on plasma sodium i.e. on increased sodium intake, sodium is added to the extracellular fluid & produces transient increases in plasma sodium, the plasma osmolality also increases stimulating osmomo receptors. It leads to stimulation of thirst and A.D.H. release, leading to expansion of plasma volume and dilution of ingested sodium. Thus over all changes in plasma sodium is small. Since plasma sodium level is not the main regulating force for aldosterone secretion, the changes in aldosterone secretion that accompany changes in sodium level must be primarily mediated through angiotension II.

The decreased renal excretion of sodium is well documented feature of post operative period. Hardey J.P.(I.S.Ravider 1952). In addition to aldosterone the functional extracellular fluid volume has recently been shown to be another major determinent of renal sodium excretion through the former in the normal individual (Spstein F.H., 1957). The decrease in functional extracellular fluid volume during the per operative period itself is a strong stimulus for aldosterone secretion, which causes sodium retention in the post operative period. Conversely a fall in plasma sodium concentration often to hyponatremic levels is also known to occur after trauma and surgical procedures (Flear C.T.G., Bhattacharya, S.C., Singh C.M. (1971) & Chan S., Redcliffe A. Johnson A. 1980) inspite of a raised aldosterone level. It is widely believed that fall in plasma sodium after an complicated surgery results from endogenous dilution. In patient who are severely ill it is after operation profound fall in plasma sodium may occur and osmolar gaps seen (Tindell S.F., Clark R.G., 1976, Flear C.T.G. Singh C.M., 1963). Part of this hyponatremia can be explained on the basis of an obligatory antidiuresis due to raised antidiuretic hormone level lasting for 24-36 hours (LeQuesne & Lewis 1952 Chan et al, 1980

post surgery. Thus the hyponatremia in post operative period is provoked by an even greater gain of water (C.M. Singh & C.T.G. Flear, 1968). Another basis of post operative hyponatremia was hypothetized by Shaires et al (1961) to be due to a significant loss of fluid with in the third space.

Another determinant of serum sodium level is the type of fluid infused in the post operative period. If a large volume of solute free fluid is given to the patient during operation or post operative period the most common electrolyte abnormality seen, following surgery is hyponatremia. The raised aldosterone and cortisol level after surgery was the basis of present concept of giving salt free fluid in early post operative periods. But during fluid planning what had not been considered, was the raised level of A.D.H., decreased real excretion of sodium and loss of extracellular fluid volume due to losses in the third space & dilutional hyponatremia post surgically. It is the balance between sodium retaining and sodium dilutional factors which ultimately determines the serum level in the post operative period.

Our study clearly shows the importance of type of fluid administration on serum sodium level other factors being identical except the amount of sodium, administration in the post operative period. There was a significant fall in serum sodium level, persisting upon to 48 hours post operatively in patients receiving 3 lit. 5% dextrose and 2 lit. of dextrose 5% + 1 lit. of isotonic saline. While there was significant rise in patients receiving 2 lit. of isotonic saline + 1 lit. of 5% dextrose The fall in serum sodium level in patient receiving 3 lit. dextrose 5%/day was significant enough to be classified as hyponatremic ($pNa^+ \angle$ 135 meq/lit.).

Our findings agree with there of A.J. Guy, J.A. Micheals and C.J.G. Flear (1987), Flear C.T.G. Bhattacharya S.S. & Singh S.M.(1971) & Chan S. Redcliffe A.R. Johnson A (1980). Their study showed that there is hyponatremia in post operative period in patients undergoing surgical procedure & receiving salt free fluid per & post operatively.

Study also includes the percentage of patient which becomes hyponatremic or shows decreased serum sodium level ($\angle$ 135 mmol/lit.) in post operative period. It was compared with the study of A.J.Guy, J.A.Micheals and C.T.G. Flear (1987) which showed that about 27.5% patients who underwent various types

of surgical procedure become hyponatremic on first post operative day while our study showed an incidence of 60% patients with 3 lit. of 5% dextrose, 37.5% receiving 2 lit. dextrose + 1 lit. isotonic saline became hyponatremic. On pre operative day in this study 20% of patient receiving 3 lit. of 5% dextrose & 12.5% of patient receiving 2 lit. 5% dextrose & 1 lit. isotonic saline are hyponatremic. While hyponatremia on first post operative day in patient receiving 3 lit. of 5% dextrose increases to 60% & patient receiving 2 lit. of 5% dextrose + 1 lit. of isotonic saline to 37.5%. On second post operative day 73.33% of group I & 43.75% of group II patient are hyponatremic.

In the third group where 33 patient received 2 lit. isotonic saline + 1 lit. dextrose 5%, 9% of patient were hyponatremic pre operatively & post operatively. Thus the percentage of patients remained constant in this group. No patient showed any clinical sign of hypernatremia namely pulmonary or peripheral edema. It can be stated that infusion of balanced salt solution to the patients undergoing surgical procedure prevents hyponatremia and quick recovery of these patients (A.J.Guy et al, 1987).

Serum potassium in relation to surgical procedure and fluid administration.

Serum potassium is usually found elevated after surgical procedures and elevation is usually dependent upon the severity of trauma or surgical procedure. In cases of major surgery, elevation is more than with minor surgical procedures.

This study shows an elevation of serum potassium on first and second post operative day. This elevation of serum potassium level is independent of nature of post operative fluid infusion (i.e. 5% dextrose) normal saline. Our study differs from study of A.J.Guy et al (1987). They study showed a fall in serum potassium level in post operative periods. But nature of fluid administration did not effect the serum potassium level. The reason for the elevated serum potassium level.

1. Cell damage & liberation of potassium from the cells.
2. Change in membrane potential so potassium passes into the extracellular fluid.
3. Acidosis

Serum osmolality in relation to surgical procedure & fluid administration.

The serum osmolality measures the total concentration of all osmotically active entities

in the plasma. Sodium is the principal cation of extracellular fluid. Increase or decrease in serum osmolality is as a consequence of increase or decrease of sodium or concentration of other osmotically active substances. The serum osmolality decreases where there is retention of fluid as a results of excess A.D.H. activity which is feature of body response to trauma causing dilutional hypo-natremia and also if there is post operative infusion of salt free fluids.

It is widely beleived that fall in plasma sodium after uncomplicated surgery results from endogenous dilution. In patient severely ill after operation profound fall in plasma sodium may occur and osmolar gaps seen (Tindell S.F., Clark R.G., 1976 & Flear C.T.G., Singh C.M. 1963). The lowering of plasma sodium may be abrupt or slow and sustained.An abrupt fall in sodium plasma are often accompanied by a reduced osmolality. The sick cell concept attributes osmolar gaps to isotonic rediistribution of solutes, from cells to extracellular fluid, caused by an abrupt increase in cell membrane permeability and the sus-tained dilution with no osmolar gaps to wide spread impaired capability of cells to maintain their normal content of non diffusable solute (Flear C.T.G., 1970, Flear C.T.G., Singh C.M. 1972, 1973).

Present study suggests a significant fall of serum osmolality in patients receiving 3 lit. of 5% dextrose while insignificant fall of serum osmolality in patient receiving 2 lit. of 5% dextrose + 1 lit. of isotonic saline post operatively. There was a slight or insignificant rise of serum osmolality in patient receiving 1 lit. of 5% dextrose + 2 lit. of isotonic saline. The above findings are because of dilutional hyponatraemia & our findings corresponds with the study of T.T. Irwin C.J. et al (1977). They also observed a significant fall in serum osmolality in patients who were kept on salt free fluid or dextrose saline solution.

Serum osmolality also depends upon blood urea & blood sugar. In our study there was no change in blood sugar level but slight fall in blood urea level but which was insignificant.

<u>Urinary volume in relation to surgical procedure and fluid administration.</u>

The present study shows an apparent increase in urine volume post operatively. The reason for increase in urine measured was because of irrigation of urinary bladder by isotonic saline in patient who underwent Frayer's modified prostectomy.

If these patients were excluded there was fall of urine output in first day in all three groups. On the IInd & subsequent post operative days there was a significant rise of urine output. Thus there is retention of water within 24-36 hours post operatively. The low urine output was seen on the day of operation in all groups i.e. patients receiving 3 lit. 5% dextrose, 2 lit. 5% dextrose + 1 lit. isotonic saline and 1 lit. 5% dextrose + 2 lit. of isotonic saline respectively. The patients receiving 3 lit. of 5% dextrose per day showed a higher urine output in comparison to patient receiving isotonic saline, urine output almost becomes similar on 3rd or 4th post operative day.

The present study shows similar results as shown by study of S.F. Tindell, R.G. Clark (1981) J.H. Thomas, D.B.Morgan (1979) and J.H. Thomas et al (1979). They observed that on the day of operation the vasopressin or antidiuretic hormone level increases to the extent that they were much higher than those achieved by simple water depletion and much higher than would be expected from alterations in plasma sodium concentration. Thus rise of serum arginine vasopressin level remains for 24 - 36 hours thereby

leading to increased water reabsorption & decreased urine formation. This initial increase in arginine vasopressin in either group was a part of stress response (Moran et al, 1964).

Moran et al (1964) have identified four phases of vasopressin secretion following surgery

I. Phase I. Normal pre-operative control period, in which plasma vasopressin concentration is within normal range.

Phase II - elevation of A.D.H. level due to overnight fast.

Phase III- It results from cutaneous and visceral stimuli and lasts from skin incision to closure.

Phase IV - Post operative phase in which there is an early increase in plasma vasopressin concen.- tration which comes to normal upto 5th post operative day.

Moran et al (1964) hypothetized that there are four affecent reflexes controlling this vasopressin release. They are

- Osmoreceptor
- Baroreceptor and
- Left atrial stretch receptor.

All of the above three receptor are negative feed back receptors.

Fourth reflex is mediated by painful stimuli which is not a feed back reflax. Therefore in presence of pain vasopressin secretion can occur even in phases of hypoosmolar hyponatremic condition. This explains a persistant secretion of vasopressin and also explains low urine output in the post operative periods.

Urinary sodium in relation to surgical procedure and fluid administration.

Our study showed a significant fall in urinary sodium in patient group receiving 3 lit. of 5% dextrose /day and 2 lit. 5% dextrose + 1 lit. of isotonic saline, while there was no significant change in the urinary sodium on the first post operative day in patients receiving 2 lit. of isotonic saline + 1 lit. of 5% dextrose. But a significant rise in urine sodium on the IInd & IIIrd post operative days. Result of our study for urinary sodium is similar to the findings of Tom Shires, Jack William M.B. & Frank Brown D. (1961) and T.T.Irvin,et al.

We have calculated for our patients the minimum requirement, to maintain water balance & to prevent fall in plasma sodium. It was seen that patients who have been infused with 5% dextrose only were hyponatremic. They had no osmotic drive for

vasopressin secretion (I.H.Thomas and D.B.Morgan, 1979. The fall in plasma sodium occured on the first post operative day in patients receiving 3 lit. of 5% dextrose. Atleast 130 mmol/lit. of sodium chloride is to be given to prevent hyponatremic Thomas and Morgan (1979) concluded from their studies that normal saline alone should be given during the early post operative period to avoid the development of hyponatremic. The present study shows that patient receiving 1 lit. of 5% dextrose + 2 lit. of isotonic saline results in maintenerme of plasma sodium at pre operative level. When planning a fluid regimen it should not be forgotton that third space losses are not treu lossex, as the fluid is reabsorved unless they are lost through wound drainage.

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CONCLUSIONS

CONCLUSIONS

Present study of 126 patient were done in M.L.B. Medical College & Hospital, Jhansi, to see the effect of type of perioperative fluid infusion in surgical patient post operatively on the following parameters.

- Serum sodium
- Serum potassium
- Serum osmolality
- Urinary volume
- Urinary sodium excretion

The patients were divided into three groups. Out of 126 patient,

- 45 received 3 lit. of 5% dextrose.
- 45 received 2 lit. of 5% dextrose + 1 lit. of isotonic saline.
- 33 received 1 lit. of 5% dextrose + 2 lit. of isotonic saline.

The conclusion derived were as follows:-

1. Serum sodium - The nature of fluid has a profound effect on serum sodium levels, salt free solution i.e. Dextrose 5% causes hyponatremia in post operative period while with balanced salt solution serum sodium levels remained within the normal range in post operative periods.

2. Serum potassium - The nature of fluid i.e. 5% dextrose or isotonic saline has no effect on serum potassium level. We found a rise of serum potassium in all three groups.

3. Serum osmolality - Patients receiving 3 lit. 5% dextrose and these receiving 2 lit. 5% dextrose + 1 lit. isotonic sailine showed a fall in serum osmolality while tere was no change or insignificant rise in serum osmolality in patient receiving 1 lit. 5% dextrose + 2 lit. isotonic saline.

4. Urine volume - Nature of fluid has an effect on the urine volume in the early post operative period. There was an increased vurine volume in patients receiving 5% dextrose only. But urine volume became equal in all groups, the second & third post operative days.

5. Urinary sodium - The salt free fluid (i.e. 5% dextrose) causes fall in urinary sodium excretion while rise is seen in patient receiving 1 lit. 5% dextrose + 2 lit. isotonic saline.

Thus, my study shows that infusion of salt free solution (i.e. 5% dextrose) in post operative period leads to hyponatremia with fall in serum osmolality and urinary sodium excretion. With infusion of balanced salt solution, there is no hyponatremia & no fall in serum osmolality, which helps in recovery of patient. Various causes are atributed to hyponatemia and fall in serum osmolality. But exact cause has not been established till now. The presence of IIIrd factor & its role require

further work.

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